	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	7710	retinoid or retinol	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:39			0
2	BRS	L2	1341	n-acetylcysteine	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:39			0
3	BRS	L3	2025	acetylcysteine	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:39			0
4	BRS	L4	2025	2 or 3	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:39			0
5	BRS	L5	59168	choleciferol or (vitamin adj K) or tocopherol or (ascorbic adj acid)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:40			0
6	BRS	L6	28	1 same 4 same 5	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:41			0
7	BRS	L7	14	((retinoid or retinol or retinamide or (retinoic adj acid)) same N-acetylcysteine) same (choleciferol or (vitamin adj K) or tocopherol or (ascorbic adj acid))	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:50			0
8	BRS	L8	214989	niacin or thiamine or riboflavin or (folic adj acid) or pyrodoxine or (pantothenic adj acid) or niacinamide or (lipoic adj acid) or (dihydrolipoic adj acid) or (amino adj acid)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:52			0

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
9	BRS	L9	619019	humectant or antioxidant or preservative or fragance or (surface adj active adj agent) or binder or (skin adj protectant adj agent)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:53			0
10	BRS	L11	3	7 same 8	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:53			0
11	BRS	L12	7	7 same 9	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 07:54			0
12	BRS	L13	7	(retinoid or retinol or retinamide or (retinoic adj acid)) same N-acetylcysteine same tocopherol same (ascorbic adj acid)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 08:01			0
13	BRS	L14	2	composition same (11 or 12 or 13)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 08:02			0
14	BRS	L15	11	kung adj john.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 08:02			0
15	BRS	L17	55	liu adj jue-chen.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 08:03			0
16	BRS	L18	1	(15 or 17) and 6	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/08/13 08:03			0

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FILE 'MEDLINE' ENTERED AT 08:18:5[redacted]N 13 AUG 2003

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FILE 'AGRICOLA' ENTERED AT 08:18:57 ON 13 AUG 2003

=> s retinoid or retinol or retinamide or (retinoic acid)
L1      172479 RETINOID OR RETINOL OR RETINAMIDE OR (RETINOIC ACID)

=> s N-acetylcysteine or acetylcysteine
L2       27985 N-ACETYLCYSTEINE OR ACETYLCYSTEINE

=> s choleciferol or (vitamin K) or tocopherol or (ascorbic acid)
L3      283346 CHOLECIFEROL OR (VITAMIN K) OR TOCOPHEROL OR (ASCORBIC ACID)

=> s l1 (p) l2 (p) l3
L4           29 L1 (P) L2 (P) L3

=> duplicate remove l4
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L4
L5           14 DUPLICATE REMOVE L4 (15 DUPLICATES REMOVED)

=> s l5 (p) composition
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L38 (P) COMPOSITI'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L40 (P) COMPOSITI'
L6            1 L5 (P) COMPOSITION

=> d l6 1 ibib abs

L6   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER:         2000:839120  CAPLUS
DOCUMENT NUMBER:          134:21446
TITLE:                    Compositions for stabilizing oxygen-labile
                          pharmaceuticals
INVENTOR(S):              Kung, John; Liu, Jue-chen
PATENT ASSIGNEE(S):       Johnson & Johnson Consumer Companies, Inc., USA
SOURCE:                   Eur. Pat. Appl., 17 pp.
                          CODEN: EPXXDW
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.   DATE
     --------------    ----  --------       --------------    --------
     EP 1055720         A2   20001129       EP 2000-304519    20000526
     EP 1055720         A3   20010307
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO
     CA 2309520         AA   20001128       CA 2000-2309520   20000525
     JP 2001011441      A2   20010116       JP 2000-158635    20000529
     CN 1284327         A    20010221       CN 2000-118833    20000529
     BR 2000003780      A    20010403       BR 2000-3780      20000616
     US 2002123460      A1   20020905       US 2001-33492     20011227
PRIORITY APPLN. INFO.:                    US 1999-136442P   P  19990528
                                          US 1999-361425    A  19990727
AB   This invention relates to   ***compns***  . and methods for stabilizing
     oxygen-labile species.  More particularly, it relates to   ***compns***  .
     contg. 1 or more oil- and/or water-sol. oxygen-labile species and one or
     more stabilizing elements.  It also relates to methods of making such
```

compns . and methods of using such ***compns*** . Thus, a formulation contained water 96, disodium EDTA 0.20, phenoxyethanol 0.73, methylparaben 0.20, propylparaben 0.07 and hydroxyethyl cellulose 1.00% for the water phase; BHT 0.10, GMS 2.00 cetearyl glucoside 3.000, C12-15 alkyl benzoate 2.00, avobenzone 2.00, octyl methoxycinnamate 4.00, and ascorbyl palmitate 0.50% for the oil phase; ***ascorbic*** ***acid*** 5.00, ***tocopherol*** 0.05, ***retinol*** 0.25, lactoferrin and thioxanthine and uric acid 1.00, ***N*** - ***acetylcysteine*** 0.01, EtOH 2.78 and 20% NaOH 9.04% as the additives. After a 13-wk incubation at 40.degree., 90% vtamin C and 96% vitamin A remained in the ***compn*** .

=> d his

(FILE 'HOME' ENTERED AT 08:18:36 ON 13 AUG 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 08:18:57 ON 13 AUG 2003

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L1       172479 S RETINOID OR RETINOL OR RETINAMIDE OR (RETINOIC ACID)
L2        27985 S N-ACETYLCYSTEINE OR ACETYLCYSTEINE
L3       283346 S CHOLECIFEROL OR (VITAMIN K) OR TOCOPHEROL OR (ASCORBIC ACID)
L4           29 S L1 (P) L2 (P) L3
L5           14 DUPLICATE REMOVE L4 (15 DUPLICATES REMOVED)
L6            1 S L5 (P) COMPOSITION

=> s l5 not l6
L7           13 L5 NOT L6

=> d l7 1-13 ibib abs

L7   ANSWER 1 OF 13     MEDLINE on STN
ACCESSION NUMBER:    2001513114      MEDLINE
DOCUMENT NUMBER:     21445113   PubMed ID: 11560978
TITLE:               Lung cancer chemoprevention: an integrated approach.
AUTHOR:              Lippman S M; Spitz M R
CORPORATE SOURCE:    Departments of Clinical Cancer Prevention, Thoracic/Head
                     and Neck Medical Oncology, and Epidemiology, University of
                     Texas M.D. Anderson Cancer Center, Houston, TX 77030, USA..
                     slippman@mdanderson.org
CONTRACT NUMBER:     CA16672 (NCI)
     CA45809 (NCI)
     CA55769 (NCI)
     CA86390 (NCI)
SOURCE:              JOURNAL OF CLINICAL ONCOLOGY, (2001 Sep 15) 19 (18 Suppl)
                     74S-82S.  Ref: 87
                     Journal code: 8309333. ISSN: 0732-183X.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
                     General Review; (REVIEW)
                     (REVIEW, TUTORIAL)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200110
ENTRY DATE:          Entered STN: 20010919
                     Last Updated on STN: 20011015
                     Entered Medline: 20011011
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AB Lung cancer is the leading cause of cancer deaths in the United States and the world, with grim incidence and mortality figures underscoring the need for new approaches, such as chemoprevention, for controlling this disease. There have been definitive, randomized, controlled lung-cancer chemoprevention trials in the three chemoprevention trial settings: primary (healthy high-risk [eg, smokers]), secondary (premalignant lesions), and tertiary (prevention of second primary tumors in previously treated patients), all of which produced negative (either neutral or harmful) primary end point results. These trials established that lung cancer was not prevented by alpha- ***tocopherol*** , beta-carotene, ***retinol*** , retinyl palmitate, ***N*** - ***acetylcysteine*** , or isotretinoin in smokers. Provocative leads of the definitive trials include the possible activity of isotretinoin in never and former smokers and that of alpha- ***tocopherol*** in prostate cancer prevention. A major area of lung cancer research is molecular epidemiologic study of highest smoking-related risk based on the interactions between tobacco carcinogens, genetic polymorphisms involved in activating and detoxifying these carcinogens, and host-cell efficiency in monitoring and repairing tobacco carcinogen-DNA damage. The future of lung cancer chemoprevention will rely heavily on molecular studies of carcinogenesis and drug

mechanisms to develop novel chemopreventive targets and drugs, risk markers, and surrogate end point biomarkers; new preclinical drug-testing models; novel imaging techniques for monitoring agent activity; and molecular epidemiologic risk models for identifying the highest-risk current and former smokers.

L7 ANSWER 2 OF 13 MEDLINE on STN
ACCESSION NUMBER: 2000222660 MEDLINE
DOCUMENT NUMBER: 20222660 PubMed ID: 10762023
TITLE: Criteria for implementation of large and multiagent clinical chemoprevention trials.
AUTHOR: Meyskens F L Jr
CORPORATE SOURCE: Department of Medicine and Biological Chemistry, Chao Family Comprehensive Cancer Center, University of California Irvine, Orange 92868, USA.. FLMeyske@UCI.edu
CONTRACT NUMBER: P30CA 62203 (NCI)
SOURCE: JOURNAL OF CELLULAR BIOCHEMISTRY. SUPPLEMENT, (2000) 34 115-20. Ref: 25
Journal code: 8207539. ISSN: 0733-1959.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000720
Last Updated on STN: 20000720
Entered Medline: 20000713

AB If one were to wait for the perfect set of experimental results before launching a multi-agent chemoprevention or large risk reduction study, the trial would never be launched. On the other hand, non-scientific considerations have led to the premature launching of at least three prominent studies (CARET, Carotene and ***Retinol*** Efficacy Trial; ATBC, Apha ***Tocopherol*** Beta Carotene; PCPT, Prostate Cancer Prevention Trial) and the much delayed start-up of another, BCPT, the Breast Cancer Prevention Trial. Strong epidemiologic data by itself should not be adequate to justify starting a large trial; experimental and/or clinical data should be developed. On the other hand fear of secondary adverse events that are of low incidence should not be enough to delay a trial if the overall health benefit could be high. The development of multiagent chemoprevention trials requires that each agent is active and additively or synergistically so in combination in preclinical models. Additionally, side effects of each agent should be non-overlapping and low to non-existent, preferably a feature determined in formal phase IIa and IIb trials. These principles will be discussed in the context of prior (CARET, ATBC) and ongoing (EUROSCAN, ***acetylcysteine*** / ***retinol***), as well as proposed future trials (difluromethyl/sulindac).

L7 ANSWER 3 OF 13 MEDLINE on STN
ACCESSION NUMBER: 2000126354 MEDLINE
DOCUMENT NUMBER: 20126354 PubMed ID: 10657911
TITLE: Lung cancer chemoprevention.
AUTHOR: Khuri F R; Lippman S M
CORPORATE SOURCE: Departments of Thoracic/Head and Neck Medical Oncology, The University of Texas M. D. Anderson Cancer Center, Houston, Texas 77030, USA.
SOURCE: SEMINARS IN SURGICAL ONCOLOGY, (2000 Mar) 18 (2) 100-5. Ref: 46
Journal code: 8503713. ISSN: 8756-0437.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000320
Last Updated on STN: 20000320
Entered Medline: 20000307

AB Lung cancer is the leading cause of cancer death in the United States. The persisting grim lung cancer incidence and mortality figures argue powerfully for new approaches such as chemoprevention for controlling this disease. ***Retinoids*** are among the most intensively studied cancer chemoprevention agents, including in the lung. Several randomized clinical or translational chemoprevention trials (e.g., of

retinoids , beta-carotene, or combined folic acid and vitamin B(12)) have been conducted for lung pre-malignancy. ***Retinoid*** studies have produced important data on molecular/cellular markers of lung carcinogenesis, e.g., loss of heterozygosity (LOH) at 3p and 9p and ***retinoic*** ***acid*** receptor-beta (RAR-beta). Two large randomized trials with a lung cancer endpoint, the Alpha- ***Tocopherol*** , Beta-Carotene (ATBC) Prevention Study and the Beta-Carotene and ***Retinol*** Efficacy Trial (CARET), found that beta-carotene (+/- ***retinol***) was harmful (in smokers). Recently completed lung-second-primary-tumor-prevention trials include the ***retinoids*** retinyl palmitate and 13-cis- ***retinoic*** ***acid*** (13cRA) and ***N*** - ***acetylcysteine*** (NAC). Vitamin E and selenium show promise for lung cancer prevention, based on positive secondary/subset analyses of three large-scale, randomized National Cancer Institute (NCI) cancer prevention trials. Future directions of lung cancer chemoprevention include the study of molecular markers of risk and drug activity, molecular targeting study, improved imaging techniques (e.g., molecular imaging) and new drug delivery systems.

L7 ANSWER 4 OF 13 MEDLINE on STN

ACCESSION NUMBER: 95111119 MEDLINE
DOCUMENT NUMBER: 95111119 PubMed ID: 7811993

TITLE: Regulation of apoptosis induced by the retinoid N-(4-hydroxyphenyl) retinamide and effect of deregulated bcl-2.

AUTHOR: Delia D; Aiello A; Formelli F; Fontanella E; Costa A; Miyashita T; Reed J C; Pierotti M A
CORPORATE SOURCE: Istituto Nazionale Tumori, European Institute of Oncology, Milan, Italy.

CONTRACT NUMBER: CA-47956 (NCI)
CA-60181 (NCI)
SOURCE: BLOOD, (1995 Jan 15) 85 (2) 359-67.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950217
Last Updated on STN: 19950217
Entered Medline: 19950203

AB The cancer chemopreventive ***retinoid*** N-(4-hydroxyphenyl)-all-trans ***retinamide*** (HPR) was recently shown by us to have antiproliferative and apoptotic effects on human leukemic cell lines, including those unresponsive to all-trans ***retinoic*** ***acid*** (ATRA). We have now characterized further the process of HPR-induced cell death. We report that inhibitors of RNA transcription and of protein synthesis, activators of protein kinase C (PKC), inhibitors of tyrosine kinases, Zn++, and the antioxidants ***acetylcysteine*** , ***ascorbic*** ***acid*** , alpha- ***tocopherol*** , and deferoxamine suppressed HPR-induced apoptosis. HL60 cells induced toward monocytic differentiation by 1,25 dihydroxyvitamin-D3 [1,25(OH)2D3], but not those induced toward the granulocytic differentiation by ATRA, showed reduced responses to HPR. The transport of HPR by cells with different sensitivity to the ***retinoid*** , however, was similar, even after treatment with the phorbol ester 12-O-tetradecanoylphorbol-13-acetate (TPA), which induces unresponsiveness to HPR. The expression of the apoptosis-related genes bcl-2, p53, and c-myc was examined to determine their role in HPR-triggered cell death. The levels of bcl-2 mRNA were markedly diminished by 24 hours of HPR treatment in all cell lines except in the relatively HPR-insensitive line K422. However, probably because of its long half-life, bcl-2 protein levels were either unchanged or only slightly decreased. Downregulation of p53 mRNA was also observed within 24 hours of HPR exposure in NB4 but not K422 cells, but no changes in the amount of p53 protein were found. Suppression of c-myc transcription was observed in all cells except K422. The protective role of bcl-2 on cell death by HPR was investigated in HL60 as well as 697 pre-B leukemia and Jurkat T-acute lymphocytic leukemia (T-ALL) cells constitutively expressing high levels of bcl-2 proteins due to gene transfer manipulation. Compared with control cells, the onset of apoptosis in these cells with deregulated bcl-2 production was delayed by at least 24 hours. These findings establish that cell death by HPR requires RNA transcription and protein synthesis and is regulated by the activation of PKC. Although changes in bcl-2, p53, and c-myc expression are found in cells treated with HPR, the time-course of these events suggests that

HPR-triggered apoptosis is not directly controlled by these genes. Finally, while ectopic overexpression of bcl-2 does not protect cells from death by HPR, it markedly delays its onset.(ABSTRACT TRUNCATED AT 400 WORDS)

L7 ANSWER 5 OF 13 MEDLINE on STN
ACCESSION NUMBER: 90199728 MEDLINE
DOCUMENT NUMBER: 90199728 PubMed ID: 2138505
TITLE: Inhibition of transformation in cultured rat tracheal epithelial cells by potential chemopreventive agents.
AUTHOR: Steele V E; Kelloff G J; Wilkinson B P; Arnold J T
CORPORATE SOURCE: Environmental Sciences Division, NSI Technology Services Corporation, Research Triangle Park, North Carolina 27709.
CONTRACT NUMBER: N01-CN55503-03 (NCI)
SOURCE: CANCER RESEARCH, (1990 Apr 1) 50 (7) 2068-74.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199004
ENTRY DATE: Entered STN: 19900601
Last Updated on STN: 19900601
Entered Medline: 19900430

AB Twenty-eight compounds were screened for chemopreventive activity by using a rat tracheal epithelial cell transformation inhibition assay. In this new assay, chemicals were tested for their ability to inhibit the formation of transformed rat tracheal epithelial cell colonies which arise following exposure to the carcinogen benzo(a)pyrene. The 15 positive compounds were ***N*** - ***acetylcysteine*** , bismuththiol, calcium glucarate, (+/-) catechin, diallyl disulfide, glycaric acid, D-glucaro-1,4-lactone, N-(4-hydroxyphenyl) ***retinamide*** , D-limonene, mesna, ***retinoic*** ***acid*** , rutin, quercetin, silymarin, and taurine. In examining the nature of compounds that inhibited rat tracheal epithelial cell transformation, several possible chemopreventive mechanisms appeared to be predominant: compounds that were positive (a) increased glutathione levels or enhanced conjugation; (b) increased cytochrome P-450 activity; (c) displayed nucleophilic activity; or (d) induced differentiation. Thirteen compounds were negative in the rat tracheal epithelial transformation inhibition assay: crocetin, difluoromethylornithine, ellagic acid, esculetin, enoxalone, ibuprofen, levamisole, nordihydroguaiaretic acid, L-2-oxothiazolidine-4-carboxylate, piroxicam, sodium butyrate, D-alpha- ***tocopherol*** acetate, and polyethylene glycol 400. It was evident from these results that this assay would not detect compounds that were (a) anti-promoting in nature; (b) glutathione inhibitors; (c) differentiation inhibitors; (d) O6-methylguanine inhibitors; (e) organ specific; or (f) inactive. The rat tracheal epithelial cell transformation inhibition assay appeared to identify chemopreventive compounds that act at early stages of the carcinogenic process.

L7 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:162784 CAPLUS
DOCUMENT NUMBER: 137:194801
TITLE: Cancer chemoprevention - present and future
AUTHOR(S): Fujiki, Hirota; Suganuma, Masami
CORPORATE SOURCE: Saitama Cancer Center, Japan
SOURCE: Biotherapy (Tokyo, Japan) (2002), 16(1), 1-9
CODEN: BITPE9; ISSN: 0914-2223
PUBLISHER: Gan to Kagaku Ryohosha
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese

AB A review with refs. The term "cancer chemoprevention", defined as prevention of the occurrence of cancer by administration of one or more compds., was coined by Michael B. Sporn in 1976. The significance of cancer chemoprevention is now internationally accepted, and interest in Japan is accelerating. This article looks at notable results of clin. trials conducted in the U.S. and Europe, and touches upon research activities in Japan. The good news includes promising results on cancer prevention in the breast, colon and liver. Main topics: 1) Breast cancer prevention trials with tamoxifen have provided effective results for individuals in the high risk group, patients with premalignant lesions-ductal carcinoma in situ (DCIS) - and contralateral breast cancer patients. 2) Primary lung cancer prevention trials with alpha- ***tocopherol*** , .beta.-carotene (ATBC) in Finland, and with .beta.-carotene and ***retinol*** (CARET) in the U.S., showed an unexpected increase in lung cancer incidence, and recently reported

results of a EUROSCAN study did not show any benefits from vitamin A and ***N*** - ***acetylcysteine*** . These three major studies indicate that a new approach is required. 3) Encouraging results with prostate cancer prevention by finasteride are anticipated. 4) The FDA has approved celecoxib, a selective Cox-2 inhibitor, for the prevention of polyp development in patients with familial adenomatous polyposis. 5) Acyclic ***retinoid*** , polyprenoic acid prevented second primary hepatomas after surgical resection of the original tumor or percutaneous injection of ethanol, mediated through clonal deletion of malignant cells in the remnant liver. 6) Finally, we discuss cancer prevention with green tea. Based on results of basic studies of (-)-epigallocatechin gallate (EGCG) and green tea polyphenols, and also results of a prospective cohort study with 8,552 individuals, we are now moving toward prevention of cancer in various organs (both primary tumor and recurrence) by introducing the "Saitama System": the equiv. of 10 Japanese-size cups of green tea per day (2.5 g green tea ext. per day) in a combination of daily beverage and green tea tablets. It is our hope that this article will provide information that will spur an increase in cancer prevention trials in Japan.

L7 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:746946 CAPLUS
DOCUMENT NUMBER: 136:47875
TITLE: Lung cancer chemoprevention: an integrated approach
AUTHOR(S): Lippman, Scott M.; Spitz, Margaret R.
CORPORATE SOURCE: Departments of Clinical Cancer Prevention, University of Texas M.D. Anderson Cancer Center, Houston, TX, 77030, USA
SOURCE: Journal of Clinical Oncology (2001), 19(18, Suppl.), 74s-82s
CODEN: JCONDN; ISSN: 0732-183X
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Lung cancer is the leading cause of cancer deaths in the United States and the world, with grim incidence and mortality figures underscoring the need for new approaches, such as chemoprevention, for controlling this disease. There have been definitive, randomized, controlled lung-cancer chemoprevention trials in the three chemoprevention trial settings: primary (healthy high-risk [eg, smokers]), secondary (premalignant lesions), and tertiary (prevention of second primary tumors in previously treated patients), all of which produced neg. (either neutral or harmful) primary end point results. These trials established that lung cancer was not prevented by alpha- ***tocopherol*** , beta-carotene, ***retinol*** , retinyl palmitate, ***N*** - ***acetylcysteine*** , or isotretinoin in smokers. Provocative leads of the definitive trials include the possible activity of isotretinoin in never and former smokers and that of alpha- ***tocopherol*** in prostate cancer prevention. A major area of lung cancer research is mol. epidemiol. study of highest smoking-related risk based on the interactions between tobacco carcinogens, genetic polymorphisms involved in activating and detoxifying these carcinogens, and host-cell efficiency in monitoring and repairing tobacco carcinogen-DNA damage. The future of lung cancer chemoprevention will rely heavily on mol. studies of carcinogenesis and drug mechanisms to develop novel chemopreventive targets and drugs, risk markers, and surrogate end point biomarkers; new preclin. drug-testing models; novel imaging techniques for monitoring agent activity; and mol. epidemiol. risk models for identifying the highest-risk current and former smokers.
REFERENCE COUNT: 87 THERE ARE 87 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 8 OF 13 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:649589 CAPLUS
DOCUMENT NUMBER: 136:67768
TITLE: Lung cancer
AUTHOR(S): Worden, Francis P.; Kalemkerian, Gregory P.
CORPORATE SOURCE: Department of Medicine, University of Michigan, Ann Arbor, MI, 48109, USA
SOURCE: Cancer Treatment and Research (2001), 106(Cancer Chemoprevention), 183-219
CODEN: CTRREP; ISSN: 0927-3042
PUBLISHER: Kluwer Academic Publishers
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Lung cancer is the result of the accumulation of numerous genetic and epigenetic defects within bronchial epithelial cells. Clin.

trials demonstrated that chemopreventive agents, such as .beta.-carotene, ***retinol*** /retinyl palmitate, 13-cis- ***retinoic*** ***acid*** , .alpha.- ***tocopherol*** , and ***N*** - ***acetylcysteine*** were ineffective in preventing lung cancer in select high-risk populations, and .beta.-carotene may even be harmful in active smokers. Increasing knowledge of the mol. events involved in lung carcinogenesis have led to the development of many strategies that specifically target derangements in signal transduction or cell cycle regulatory pathways.
REFERENCE COUNT: 189 THERE ARE 189 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 9 OF 13 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:482634 CAPLUS
DOCUMENT NUMBER: 135:298715
TITLE: Effects of antioxidant vitamins on anti-IgE-induced mediator release from human basophils
AUTHOR(S): Strenzke, N.; Grabbe, J.; Plath, K. E. S.; Wolff, H. H.; Gibbs, B. F.
CORPORATE SOURCE: Department of Dermatology, Medical University of Lubeck, Lubeck, D-23538, Germany
SOURCE: Inflammation Research (2001), 50(Suppl. 2), S49-S50
CODEN: INREFB; ISSN: 1023-3830
PUBLISHER: Birkhaeuser Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English
AB It was examd. whether antioxidants may provide beneficial effects by inhibiting IgE-dependent basophil mediator release. ***Ascorbic*** ***acid*** , ***N*** - ***acetylcysteine*** and glutathione were dissolved in RPMI 1640 medium and the pH was cor. to 7.4. Glutathione and its precursor ***N*** - ***acetylcysteine*** had no influence on IgE-dependent histamine release from human basophils, while ambroxol was shown to be a highly efficacious inhibitor. All the vitamins tested showed no effect on histamine release in the absence of anti-IgE. In the histamine release expts., ambroxol was the only effective inhibitor of interleukin (IL)-4 and IL-13 releases. Compared to solvent controls, ***N*** - ***acetylcysteine*** , ***retinoic*** ***acid*** and .beta.-carotene did not considerably alter anti-IgE induced cytokine releases. ***Ascorbic*** ***acid*** strongly enhanced IL-13 release, which was not significant because of the donor variation in IL-13 release. A major role for reactive oxygen species in the IgE-dependent activation of human basophils was not supported by the resulting data.
REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 10 OF 13 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1998:497254 CAPLUS
DOCUMENT NUMBER: 129:230153
TITLE: Dehydroepiandrosterone synergizes with antioxidant supplements for immune restoration in old as well as retrovirus-infected mice
AUTHOR(S): Jiang, Shuguang; Lee, Jeongmin; Zhang, Zhen; Inserra, Paula; Solkoff, David; Watson, Ronald R.
CORPORATE SOURCE: Arizona Prevention Center, Univ. Arizona, Tucson, AZ, 85724, USA
SOURCE: Journal of Nutritional Biochemistry (1998), 9(7), 362-369
CODEN: JNBIEL; ISSN: 0955-2863
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Prodn. of the antioxidant hormone dehydroepiandrosterone (DHEA) declines as immunosenescence develops in the elderly. Very old C57BL/6 female mice (29 mo), survivors after 71% had died due to aging, were evaluated after DHEA sulfate 0.01% addn. to the drinking water for 6 wk. DHEA increased the T-cell proliferation, restored secretion of Th1 cytokines (interleukin IL-2), decreased interferon-.gamma. (IFN-.gamma.) prodn., and normalized (lowered) the Th2 cytokine (IL-4 and IL-6) secretion. The survival was increased in the 29-mo-old mice treated by DHEA. DHEA sulfate, the storage form of DHEA, decreased the immune dysfunctions caused by increased oxidn. during LP-BM5 murine leukemia retrovirus infection. To verify the synergistic effect of DHEA + antioxidant nutrients, 17-mo-old mice were fed with antioxidants (mixt. of coenzyme Q10, d-.alpha.- ***tocopherol*** , L- ***ascorbic*** ***acid*** , L-carnitine, ***N*** - ***acetylcysteine*** , ***retinol*** , Se, Mg, Zn) or antioxidants + DHEA sulfate for 16 wk. DHEA sulfate + antioxidants increased the B-cell proliferation and IL-2 secretion and maintained the

Th2 cytokine secretion and hepatic vitamin E levels close to the levels seen in old noninfected mice. Vitamin did antioxidant supplementation alone. Thus, DHEA alone, and esp. DHEA sulfate plus antioxidant nutrients, can prevent immune dysfunctions in very old and in old retrovirus-infected mice.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 11 OF 13 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1998:55925 BIOSIS
DOCUMENT NUMBER: PREV199800055925
TITLE: Chemoprevention of aerodigestive cancer.
AUTHOR(S): Berwick, Marianne (1); Schantz, Stimson
CORPORATE SOURCE: (1) Dep. Epidemiology Biostatistics, Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021 USA
SOURCE: Cancer and Metastasis Reviews, (1997) Vol. 16, No. 3, pp. 329-347.
ISSN: 0167-7659.
DOCUMENT TYPE: General Review
LANGUAGE: English

L7 ANSWER 12 OF 13 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1993:421810 BIOSIS
DOCUMENT NUMBER: PREV199345069435
TITLE: Cancer prevention research trials.
AUTHOR(S): Greenwald, Peter (1); Malone, Winfred F. (1); Cerny, Mary E.; Stern, Harriet R.
CORPORATE SOURCE: (1) Div. Cancer Prevention and Control, Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20892 USA
SOURCE: Vande Woude, G. F. [Editor]; Klein, G. [Editor]. Advances in Cancer Research, (1993) Vol. 61, pp. 1-23. Advances in Cancer Research.
Publisher: Academic Press, Inc. 1250 Sixth Ave., San Diego, California 92101, USA.
ISSN: 0065-230X. ISBN: 0-12-006661-0.
DOCUMENT TYPE: General Review
LANGUAGE: English

L7 ANSWER 13 OF 13 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 2000118551 EMBASE
TITLE: Criteria for implementation of large and multiagent clinical chemoprevention trials.
AUTHOR: Meyskens F.L. Jr.
CORPORATE SOURCE: F.L. Meyskens Jr., Medicine and Biological Chemistry, Chao Family Comprehensive Can. Ctr., University of California, Orange, CA 92868, United States. FLMeyske@UCI.edu
SOURCE: Journal of Cellular Biochemistry, (2000) 77/SUPPL. 34 (115-120).
Refs: 24
ISSN: 0730-2312 CODEN: JCEBD5
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
017 Public Health, Social Medicine and Epidemiology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB If one were to wait for the perfect set of experimental results before launching a multi-agent chemoprevention or large risk reduction study, the trial would never be launched. On the other hand, non-scientific considerations have led to the premature launching of at least three prominent studies (CARET, Carotene and ***Retinol*** Efficacy Trial; ATBC, Apha ***Tocopherol*** Beta Carotene; PCPT, Prostate Cancer Prevention Trial) and the much delayed start-up of another, BCPT, the Breast Cancer Prevention Trial. Strong epidemiologic data by itself should not be adequate to justify starting a large trial; experimental and/or clinical data should be developed. On the other hand fear of secondary adverse events that are of low incidence should not be enough to delay a trial if the overall health benefit could be high. The development of multiagent chemoprevention trials requires that each agent is active and additively or synergistically so in combination in preclinical models. Additionally, side effects of each agent should be non-overlapping and low to non-existent, preferably a feature determined in formal phase IIa and IIb trials. These principles will be discussed in the context of prior (CARET, ATBC) and ongoing (EUROSCAN, ***acetylcysteine*** / ***retinol***), as well as proposed future trials (difluromethyl/sulindac). (C) 2000 Wiley-Liss, Inc.

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=> d his

     (FILE 'HOME' ENTERED AT 08:18:36 ON 13 AUG 2003)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     08:18:57 ON 13 AUG 2003
L1       172479 S RETINOID OR RETINOL OR RETINAMIDE OR (RETINOIC ACID)
L2        27985 S N-ACETYLCYSTEINE OR ACETYLCYSTEINE
L3       283346 S CHOLECIFEROL OR (VITAMIN K) OR TOCOPHEROL OR (ASCORBIC ACID)
L4           29 S L1 (P) L2 (P) L3
L5           14 DUPLICATE REMOVE L4 (15 DUPLICATES REMOVED)
L6            1 S L5 (P) COMPOSITION
L7           13 S L5 NOT L6

=> s l1 (p) l2 (p) (ascorbic acid) (p) tocopherol
L8            8 L1 (P) L2 (P) (ASCORBIC ACID) (P) TOCOPHEROL

=> duplicate remove l8
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L8
L9            4 DUPLICATE REMOVE L8 (4 DUPLICATES REMOVED)

=> d l9 1-4 ibib abs

L9   ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER:         2000:839120  CAPLUS
DOCUMENT NUMBER:          134:21446
TITLE:                    Compositions for stabilizing oxygen-labile
                          pharmaceuticals
INVENTOR(S):              Kung, John; Liu, Jue-chen
PATENT ASSIGNEE(S):       Johnson & Johnson Consumer Companies, Inc., USA
SOURCE:                   Eur. Pat. Appl., 17 pp.
                          CODEN: EPXXDW
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     EP 1055720         A2   20001129       EP 2000-304519   20000526
     EP 1055720         A3   20010307
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO
     CA 2309520         AA   20001128       CA 2000-2309520  20000525
     JP 2001011441      A2   20010116       JP 2000-158635   20000529
     CN 1284327         A    20010221       CN 2000-118833   20000529
     BR 2000003780      A    20010403       BR 2000-3780     20000616
     US 2002123460      A1   20020905       US 2001-33492    20011227
PRIORITY APPLN. INFO.:                   US 1999-136442P  P  19990528
                                         US 1999-361425   A  19990727
AB   This invention relates to compns. and methods for stabilizing
     oxygen-labile species.  More particularly, it relates to compns. contg. 1
     or more oil- and/or water-sol. oxygen-labile species and one or more
     stabilizing elements.  It also relates to methods of making such compns.
     and methods of using such compns.  Thus, a formulation contained water
     73.96, disodium EDTA 0.20, phenoxyethanol 0.73, methylparaben 0.20,
     propylparaben 0.07 and hydroxyethyl cellulose 1.00% for the water phase;
     BHT 0.10, GMS 2.00 cetearyl glucoside 3.000, C12-15 alkyl benzoate 2.00,
     avobenzone 2.00, octyl methoxycinnamate 4.00, and ascorbyl palmitate 0.50%
     for the oil phase;  ***ascorbic***  ***acid***  5.00,
     ***tocopherol***  0.05,  ***retinol***  0.25, lactoferrin and
     thioxanthine and uric acid 1.00,  ***N***  -  ***acetylcysteine***
     0.01, EtOH 2.78 and 20% NaOH 9.04% as the additives.  After a 13-wk
     incubation at 40.degree., 90% vtamin C and 96% vitamin A remained in the
     compn.

L9   ANSWER 2 OF 4  CAPLUS  COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER:         1998:497254  CAPLUS
DOCUMENT NUMBER:          129:230153
TITLE:                    Dehydroepiandrosterone synergizes with antioxidant
                          supplements for immune restoration in old as well as
                          retrovirus-infected mice
AUTHOR(S):                Jiang, Shuguang; Lee, Jeongmin; Zhang, Zhen; Inserra,
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Paula; Solkoff, David; Watson, Ronald R.
CORPORATE SOURCE: Arizona Prevention Center, Univ. Arizona, Tucson, AZ, 85724, USA
SOURCE: Journal of Nutritional Biochemistry (1998), 9(7), 362-369
CODEN: JNBIEL; ISSN: 0955-2863
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Prodn. of the antioxidant hormone dehydroepiandrosterone (DHEA) declines as immunosenescence develops in the elderly. Very old C57BL/6 female mice (29 mo), survivors after 71% had died due to aging, were evaluated after DHEA sulfate 0.01% addn. to the drinking water for 6 wk. DHEA increased the T-cell proliferation, restored secretion of Th1 cytokines (interleukin IL-2), decreased interferon-.gamma. (IFN-.gamma.) prodn., and normalized (lowered) the Th2 cytokine (IL-4 and IL-6) secretion. The survival was increased in the 29-mo-old mice treated by DHEA. DHEA sulfate, the storage form of DHEA, decreased the immune dysfunctions caused by increased oxidn. during LP-BM5 murine leukemia retrovirus infection. To verify the synergistic effect of DHEA + antioxidant nutrients, 17-mo-old mice were fed with antioxidants (mixt. of coenzyme Q10, d-.alpha.-***tocopherol*** , L- ***ascorbic*** ***acid*** , L-carnitine, ***N*** - ***acetylcysteine*** , ***retinol*** , Se, Mg, Zn) or antioxidants + DHEA sulfate for 16 wk. DHEA sulfate + antioxidants increased the B-cell proliferation and IL-2 secretion and maintained the Th2 cytokine secretion and hepatic vitamin E levels close to the levels seen in old noninfected mice than did antioxidant supplementation alone. Thus, DHEA alone, and esp. DHEA sulfate plus antioxidant nutrients, can prevent immune dysfunctions in very old and in old retrovirus-infected mice.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 4 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 95111119 MEDLINE
DOCUMENT NUMBER: 95111119 PubMed ID: 7811993
TITLE: Regulation of apoptosis induced by the retinoid N-(4-hydroxyphenyl) retinamide and effect of deregulated bcl-2.
AUTHOR: Delia D; Aiello A; Formelli F; Fontanella E; Costa A; Miyashita T; Reed J C; Pierotti M A
CORPORATE SOURCE: Istituto Nazionale Tumori, European Institute of Oncology, Milan, Italy.
CONTRACT NUMBER: CA-47956 (NCI)
CA-60181 (NCI)
SOURCE: BLOOD, (1995 Jan 15) 85 (2) 359-67.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950217
Last Updated on STN: 19950217
Entered Medline: 19950203

AB The cancer chemopreventive ***retinoid*** N-(4-hydroxyphenyl)-all-trans ***retinamide*** (HPR) was recently shown by us to have antiproliferative and apoptotic effects on human leukemic cell lines, including those unresponsive to all-trans ***retinoic*** ***acid*** (ATRA). We have now characterized further the process of HPR-induced cell death. We report that inhibitors of RNA transcription and of protein synthesis, activators of protein kinase C (PKC), inhibitors of tyrosine kinases, Zn++, and the antioxidants ***acetylcysteine*** , ***ascorbic*** ***acid*** , alpha- ***tocopherol*** , and deferoxamine suppressed HPR-induced apoptosis. HL60 cells induced toward monocytic differentiation by 1,25 dihydroxyvitamin-D3 [1,25(OH)2D3], but not those induced toward the granulocytic differentiation by ATRA, showed reduced responses to HPR. The transport of HPR by cells with different sensitivity to the ***retinoid*** , however, was similar, even after treatment with the phorbol ester 12-O-tetradecanoylphorbol-13-acetate (TPA), which induces unresponsiveness to HPR. The expression of the apoptosis-related genes bcl-2, p53, and c-myc was examined to determine their role in HPR-triggered cell death. The levels of bcl-2 mRNA were markedly diminished by 24 hours of HPR treatment in all cell lines except in the relatively HPR-insensitive line K422. However, probably because of its long half-life, bcl-2 protein levels were either unchanged or only slightly decreased. Downregulation of p53 mRNA was also observed within

24 hours of HPR exposure in [redacted] but not K422 cells, but no changes in the amount of p53 protein were found. Suppression of c-myc transcription was observed in all cells except K422. The protective role of bcl-2 on cell death by HPR was investigated in HL60 as well as 697 pre-B leukemia and Jurkat T-acute lymphocytic leukemia (T-ALL) cells constitutively expressing high levels of bcl-2 proteins due to gene transfer manipulation. Compared with control cells, the onset of apoptosis in these cells with deregulated bcl-2 production was delayed by at least 24 hours. These findings establish that cell death by HPR requires RNA transcription and protein synthesis and is regulated by the activation of PKC. Although changes in bcl-2, p53, and c-myc expression are found in cells treated with HPR, the time-course of these events suggests that HPR-triggered apoptosis is not directly controlled by these genes. Finally, while ectopic overexpression of bcl-2 does not protect cells from death by HPR, it markedly delays its onset.(ABSTRACT TRUNCATED AT 400 WORDS)

L9 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1993:421810 BIOSIS
DOCUMENT NUMBER: PREV199345069435
TITLE: Cancer prevention research trials.
AUTHOR(S): Greenwald, Peter (1); Malone, Winfred F. (1); Cerny, Mary E.; Stern, Harriet R.
CORPORATE SOURCE: (1) Div. Cancer Prevention and Control, Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20892 USA
SOURCE: Vande Woude, G. F. [Editor]; Klein, G. [Editor]. Advances in Cancer Research, (1993) Vol. 61, pp. 1-23. Advances in Cancer Research.
Publisher: Academic Press, Inc. 1250 Sixth Ave., San Diego, California 92101, USA.
ISSN: 0065-230X. ISBN: 0-12-006661-0.
DOCUMENT TYPE: General Review
LANGUAGE: English

=> d his

(FILE 'HOME' ENTERED AT 08:18:36 ON 13 AUG 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 08:18:57 ON 13 AUG 2003
L1 172479 S RETINOID OR RETINOL OR RETINAMIDE OR (RETINOIC ACID)
L2 27985 S N-ACETYLCYSTEINE OR ACETYLCYSTEINE
L3 283346 S CHOLECIFEROL OR (VITAMIN K) OR TOCOPHEROL OR (ASCORBIC ACID)
L4 29 S L1 (P) L2 (P) L3
L5 14 DUPLICATE REMOVE L4 (15 DUPLICATES REMOVED)
L6 1 S L5 (P) COMPOSITION
L7 13 S L5 NOT L6
L8 8 S L1 (P) L2 (P) (ASCORBIC ACID) (P) TOCOPHEROL
L9 4 DUPLICATE REMOVE L8 (4 DUPLICATES REMOVED)

=> s niacin or thiamine or riboflavin or (folic acid) or pyrodoxine or (pantothenic acid) or (lipo
3 FILES SEARCHED...
L10 2171983 NIACIN OR THIAMINE OR RIBOFLAVIN OR (FOLIC ACID) OR PYRODOXINE OR (PANTOTHENIC ACID) OR (LIPOIC ACID) OR (DIHYDROLIPOIC ACID) OR (AMINO ACID)

=> s humectant or antioxidant or preservative or fragrance or (surface active agent) or binder or
L11 582049 HUMECTANT OR ANTIOXIDANT OR PRESERVATIVE OR FRAGRANCE OR (SURFAC E ACTIVE AGENT) OR BINDER OR (SKIN PROTECTANT AGENT)

=> s l5 (p) (l10 or l11)
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L86 (P) '
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L88 (P) '
L12 6 L5 (P) (L10 OR L11)

=> d his

(FILE 'HOME' ENTERED AT 08:18:36 ON 13 AUG 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 08:18:57 ON 13 AUG 2003
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L2 27985 S N-ACETYLCYSTEINE OR ACETYLCYSTEINE
L3 283346 S CHOLECIFEROL OR (VITAMIN K) OR TOCOPHEROL OR (ASCORBIC ACID)

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L4            29 S L1 (P) L2 (P) L3
L5            14 DUPLICATE REMOVE L4 (15 DUPLICATES REMOVED)
L6             1 S L5 (P) COMPOSITION
L7            13 S L5 NOT L6
L8             8 S L1 (P) L2 (P) (ASCORBIC ACID) (P) TOCOPHEROL
L9             4 DUPLICATE REMOVE L8 (4 DUPLICATES REMOVED)
L10      2171983 S NIACIN OR THIAMINE OR RIBOFLAVIN OR (FOLIC ACID) OR PYRODOXIN
L11       582049 S HUMECTANT OR ANTIOXIDANT OR PRESERVATIVE OR FRAGRANCE OR (SUR
L12            6 S L5 (P) (L10 OR L11)

=> skung john/au
SKUNG IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s kung john/au
L13           10 KUNG JOHN/AU

=> s liu jue-chen/au
L14           71 LIU JUE-CHEN/AU

=> s (l13 or l14) and l1
L15           17 (L13 OR L14) AND L1

=> duplicate remove l15
DUPLICATE PREFERENCE IS 'CAPLUS, BIOSIS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L15
L16           17 DUPLICATE REMOVE L15 (0 DUPLICATES REMOVED)

=> s l16 and l2 and l3
L17            1 L16 AND L2 AND L3

=> d l17 1 ibib abs

L17  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER:         2000:839120  CAPLUS
DOCUMENT NUMBER:          134:21446
TITLE:                    Compositions for stabilizing oxygen-labile
                          pharmaceuticals
INVENTOR(S):                ***Kung, John***  ;   ***Liu, Jue-chen***
PATENT ASSIGNEE(S):       Johnson & Johnson Consumer Companies, Inc., USA
SOURCE:                   Eur. Pat. Appl., 17 pp.
                          CODEN: EPXXDW
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     EP 1055720         A2   20001129       EP 2000-304519   20000526
     EP 1055720         A3   20010307
        R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
            IE, SI, LT, LV, FI, RO
     CA 2309520         AA   20001128       CA 2000-2309520  20000525
     JP 2001011441      A2   20010116       JP 2000-158635   20000529
     CN 1284327         A    20010221       CN 2000-118833   20000529
     BR 2000003780      A    20010403       BR 2000-3780     20000616
     US 2002123460      A1   20020905       US 2001-33492    20011227
PRIORITY APPLN. INFO.:                    US 1999-136442P  P  19990528
                                          US 1999-361425   A  19990727
AB   This invention relates to compns. and methods for stabilizing
     oxygen-labile species.  More particularly, it relates to compns. contg. 1
     or more oil- and/or water-sol. oxygen-labile species and one or more
     stabilizing elements.  It also relates to methods of making such compns.
     and methods of using such compns.  Thus, a formulation contained water
     73.96, disodium EDTA 0.20, phenoxyethanol 0.73, methylparaben 0.20,
     propylparaben 0.07 and hydroxyethyl cellulose 1.00% for the water phase;
     BHT 0.10, GMS 2.00 cetearyl glucoside 3.000, C12-15 alkyl benzoate 2.00,
     avobenzone 2.00, octyl methoxycinnamate 4.00, and ascorbyl palmitate 0.50%
     for the oil phase;   ***ascorbic***     ***acid***   5.00,
       ***tocopherol***   0.05,   ***retinol***   0.25, lactoferrin and
     thioxanthine and uric acid 1.00,   ***N***  -  ***acetylcysteine***
     0.01, EtOH 2.78 and 20% NaOH 9.04% as the additives.  After a 13-wk
     incubation at 40.degree., 90% vtamin C and 96% vitamin A remained in the
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compn.

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(FILE 'HOME' ENTERED AT 08:18:36 ON 13 AUG 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 08:18:57 ON 13 AUG 2003

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L2         27985 S N-ACETYLCYSTEINE OR ACETYLCYSTEINE
L3        283346 S CHOLECIFEROL OR (VITAMIN K) OR TOCOPHEROL OR (ASCORBIC ACID)
L4            29 S L1 (P) L2 (P) L3
L5            14 DUPLICATE REMOVE L4 (15 DUPLICATES REMOVED)
L6             1 S L5 (P) COMPOSITION
L7            13 S L5 NOT L6
L8             8 S L1 (P) L2 (P) (ASCORBIC ACID) (P) TOCOPHEROL
L9             4 DUPLICATE REMOVE L8 (4 DUPLICATES REMOVED)
L10      2171983 S NIACIN OR THIAMINE OR RIBOFLAVIN OR (FOLIC ACID) OR PYRODOXIN
L11       582049 S HUMECTANT OR ANTIOXIDANT OR PRESERVATIVE OR FRAGRANCE OR (SUR
L12            6 S L5 (P) (L10 OR L11)
L13           10 S KUNG JOHN/AU
L14           71 S LIU JUE-CHEN/AU
L15           17 S (L13 OR L14) AND L1
L16           17 DUPLICATE REMOVE L15 (0 DUPLICATES REMOVED)
L17            1 S L16 AND L2 AND L3
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=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	131.06	131.27

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-5.86	-5.86

STN INTERNATIONAL LOGOFF AT 08:35:40 ON 13 AUG 2003